

Item 77 : Angina and pharyngitis in children and adults

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2013

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Objectifs ENC

- How to diagnose angina and rhinopharyngitis.
- How to diagnose infectious mononucleosis.
- Discuss the therapeutic approach (P) and plan patient monitoring.
- Know the clinical picture of acute rhinopharyngitis. Understand the risks of nasopharyngeal and sinus infections.
- Know when to evoke chronic adenoiditis.
- Know how to diagnose a peritonsillar phlegmon.
- Know the clinical picture of a peritonsillar phlegmon, differential diagnosis, and general and local treatment options.
- Know how to discuss the indications of tonsillectomy in both children and adults, and understand the contraindications and complications.

Introduction

This chapter covers :

- rhinopharyngitis (also known as adenoiditis), which constitutes the majority of nasopharyngeal pathologies encountered in children;
- other rare nasopharyngeal conditions in children are evoked as differential diagnosis;
- acute (angina) and chronic (chronic tonsillitis) tonsillar infections that occur at any age.

All of these reactionary and infectious manifestations are disorders of Waldeyer's tonsillar ring (or Waldeyer's lymphatic ring), located at the entrance of the upper aerodigestive tract. It represents an important part of the peripheral lymphoid system, along with the lymph nodes, spleen and lymphoid formations of the digestive tract. It consists essentially of (**Fig 1.**) :

- palatine tonsils, located at the isthmus faucium;
- pharyngeal tonsils (adenoids), located in the nasopharynx area;
- lingual tonsils, located at the basis of the tongue.

Fig 1.

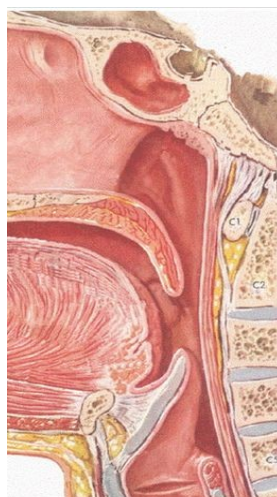
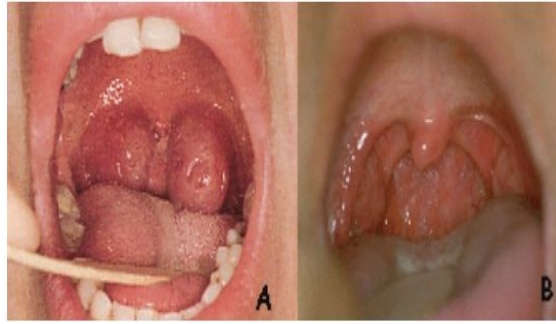


Fig 2.



1. Pathophysiology - General information

At birth, children possess only maternal immunoglobulins G (IgG) as humoral anti-infectious immune defense system: this defense system is passive and temporary (lasting approximately 6 months). During this period, the child develops its own means of immune system acquisition, namely its own lymphoid tissue.

The antigens required for this immune system synthesis enter the body through the nasal orifices and first come into contact with the mucosal lining of the nasopharynx, resulting in the development of the pharyngeal tonsil, and then secondly, with the oropharynx (palatine tonsils), and finally with the digestive tract (Peyer plates).

After passing through the mucosa, viral or bacterial antigens are captured by macrophages and transported to germinal centers of lymphoid tissue that are immune synthesis centers (due to B- and T-lymphocytes), which thus multiply, increase in volume, and result in pharyngeal tonsil hypertrophy, *i.e.*, adenoid vegetations.

Adenoidal hypertrophy (as well as tonsillar hypertrophy) must thus not be seen as a pathological manifestation, but rather as a normal reaction of an organism undergoing immune maturation.

Inflammation of the nasopharynx (rhinopharyngitis) in children represents a natural adaptation to the microbial world, and one must consider a frequency of four to five trivial rhinopharyngeal infections per annum as normal, up to the age of 6 to 7 years. This is the period within which children acquire immunity, and as such, this condition is an ailment of the adaptation process.

However, rhinopharyngitis or angina in children is considered as truly pathological when infection occurs too often or is associated with complications.

2. Rhinopharyngitis

Rhinopharyngitis is the most frequently encountered infectious disease in children and is the leading cause of pediatric consultation.

Its incidence is higher in children, particularly preschoolers, than in adults.

It is defined as an inflammatory disorder of the upper pharyngeal region (nasopharynx) that is variably associated with nasal impairment.

Rhinopharyngitis, mainly of viral origin, is a benign disease, with spontaneous improvement seen in 7-10 days.

2.1. Etiologies

Viruses are, by far, the major causal pathogens of rhinopharyngitis, including rhinovirus, coronavirus, respiratory syncytial virus (RSV), *influenza virus* and *para-influenza virus*, adenovirus, enterovirus, etc. More than 200 viruses are likely to induce rhinopharyngitis, which can be accompanied by clinical symptoms, reflecting the affliction of another part of the respiratory tract.

These viruses induce a local short-term immunity that does not protect against heterologous-type viruses

and therefore, allows re-infection to occur. The number of causal viruses, infection or re-infection status, as well as patient age account for the variability of the clinical picture. Infectivity is high for all of these viruses, especially for rhinovirus, RSV, and influenza virus.

Bacteria found in nasopharyngeal secretions (notably *S. pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, and *Staphylococcus*) are part of the commensal flora of the nasopharynx of the child. The same bacteria species are found in healthy children and those with a rhinopharyngitis.

2.2. Diagnosis

A diagnosis is easily established in a child aged from 6 months to 8 years, with a brutal infectious syndrome combining:

- fever at 38.5-39°C, sometimes higher at 40°C, mainly in the morning, with agitation and, in some cases, vomiting and diarrhea;
- nasal obstruction with mucopurulent rhinorrhea, potentially resulting in severe feeding disorders in infants;
- acute tubal obstruction with slight transmission deafness;
- painful bilateral cervical adenopathies.

Increased mucus secretions are visible on examination of the nose, and flowing on the posterior wall of the pharynx is visible on oral examination. The eardrums are congested.

Clinical examination, which is not very helpful, is aimed at eliminating another infectious site in the context of childhood fever (meningeal, joints, digestive, lung, urinary, otitis, and angina).

2.3. Treatment

Antibiotic treatment for acute uncomplicated rhinopharyngitis is not justified in either adults or children. Its effectiveness has not been demonstrated, as based on the duration of symptoms or prevention of complications, even in the presence of risk factors.

In case of uncomplicated rhinopharyngitis, symptomatic treatment (antipyretics, nose blowing, decongestants, and local antiseptics) should be implemented, and it is appropriate to inform parents/patients on the viral nature of the pathology, average duration of symptoms (7-10 days), usually spontaneously favorable progression, but also on possible complications along with their respective symptoms. Antibiotic treatment is only justified in cases of complications, most likely of bacterial origin, such as acute otitis media and sinusitis. Antibiotic treatment is not justified for preventing these complications.

2.4. Complications associated with rhinopharyngitis

The occurrence of bacterial complications justifies the prescription of antibiotics :

- acute otitis media (AOM) often occurs at early onset and is commonly seen in children aged between 6 months and 2 years;
- sinusitis:
 - from an early age, acute ethmoid sinusitis,
 - later and mostly after the age of 6, maxillary sinusitis.
- and incidentally, the following complications:
 - ganglion: cervical adenophlegmon, retropharyngeal abscess, and torticollis,
 - laryngeal: subglottic acute laryngitis and pseudocroup,
 - Digestive: diarrhea, vomiting, and dehydration of the infant,
 - hyperthermia: febrile seizures, etc.

The occurrence of a lower respiratory infection, such as a bronchitis, bronchiolitis or pneumonia, is not considered to be a complication or secondary infection of rhinopharyngitis (in this case, rhinopharyngitis is rather a prodroma or associated symptom).

The purulent features of rhinorrhea and presence of a fever (within the normal time limits of rhinopharyngitis) are not risk factors for developing complications.

2.5. Differential diagnosis

Differential diagnosis is rarely an issue.

- In the event of rhinorrhea, simple rhinitis can lead to confusion. Rhinorrhea is usually associated with rhinopharyngitis and is treated using a similar therapeutic approach.
- In the event of nasal obstruction :
 - Bilateral choanal imperforation manifests itself as a total neonatal nasal obstruction, with dramatic symptoms in newborns who cannot breathe through their mouth: asphyxia and feeding problems with false passages. Diagnosis is easy and can be made simply using a mirror placed in front of the nostrils (lack of condensation when breathing out) and a catheter introduced into both nasal cavities, which comes to a halt after a few centimeters, without passing into the pharynx. The immediate gesture is to install a Mayo tube. Alimentation is supplied by a feeding tube; surgical treatment should be performed as early as possible, consisting of perforation of the mucosal or osseous diaphragms blocking the posterior apertures of the nose;
 - Unilateral choanal imperforation does not cause any significant problems. Detection of the condition is often late, when confronted with nasal obstruction and long-term unilateral mucosal rhinorrhea. Surgical treatment can be postponed;
 - benign tumor: nasopharyngeal fibroma or male puberty bleeding fibroma. This rare, histologically benign tumor, is a highly vascularized fibromyxoma that develops at the level of the external wall of the choanal orifice. In a pubescent teenager, its gradual expansion into the nasal cavity and nasopharynx leads to progressive nasal obstruction, with rhinorrhea and recurrent epistaxis, which is more and more abundant and sometimes dramatic;
 - malignant tumors: nasopharynx cancer. Not exceptional in children.

Fig 3.

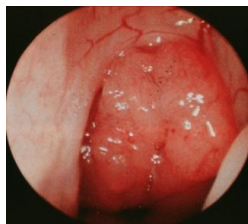


Fig 4.



2.6. Hypertrophy of adenoid vegetations causing recurrent rhinopharyngitis

The hypertrophy of the pharyngeal tonsil is considered a normal reaction during immune maturation. In case of significant enlargement, this hypertrophy may have clinical consequences. Given this scenario, there are the telltale signs of upper respiratory obstruction (nasopharyngeal):

- general appearance of the child, often pale, hypotrophic or sometimes overweight and listless;
- permanent nasal obstruction;
- breathing through the mouth;
- snoring at night with restless sleep;
- nasal voice (closed rhinolalia);
- particular facies, known as "adenoid", which is common to any chronic nasopharyngeal obstruction: mouth open with incisor open bite, dazed look, long and narrow face, and high arched palate;
- pectus carinatum (pigeon breast).

Clinical examination reveals :

- rarely an anterior bulging of the soft palate on examination of the buccal cavity, or rather the appearance of the lower part of large vegetations, especially during a gag reflex;
- cervical bilateral polyadenopathy on palpation of the neck: the lymph nodes are small (<1.5 cm), firm, and painless.

Further investigations may include :

- posterior rhinoscopy with a mirror or optical fibers for ENT investigations, which is often difficult or impractical in the young;
- nasal fibroscopy by an ENT specialist;
- lateral x-ray of nasopharynx.

Disease evolution is often beset with outbreaks of rhinopharyngitis along with possible complications.

Adenoid vegetations, which reach maximum development between 4 and 7 years, generally involute spontaneously around the age of puberty. Residual traces of adenoid vegetations may persist and cause rhinopharyngitis in adults.

Specific surgical treatment is at times necessary when :

- volume of these adenoid vegetations results in significant and permanent mechanical difficulties in breathing;
- infectious outbreaks accompanied by ear infections (with hearing loss), laryngitis, or tracheobronchitis are common.

Removal of the adenoid vegetations or adenoidectomy is a benign operation that is possible from the age of 1 year onwards, sometimes even earlier. This intervention, however, never involves total eradication of nasopharyngeal lymphoid tissue, which may recur quickly, especially in young children (recommendations of the AFSSAPS [French Health Products Safety Agency]).

2.7. Other factors favoring nasopharyngeal infections

Recurrent uncomplicated rhinopharyngitis is characterized by its subacute or chronic evolution that extends for weeks and months. These children, usually with adenoid vegetations, have a "perpetual cold", which is barely relieved during the summer months and hence, a difficult therapeutic problem. The contributing factors are :

- hypertrophy of adenoid vegetations,
- climate-related factors: spring and fall,
- epidemic factors: flu, etc.,
- lifestyle: crèche, school, "infecting" family environment, and passive smoking,
- eruptive fevers in childhood: measles, chicken pox, and scarlet fever, etc.,
- environment, family history of "mucosal fragility", allergic or otherwise.

The treatment of each factor enables recurrent rhinopharyngitis to be controlled :

- stopping of passive smoking, removal of the child from group activities if possible, and showing the child how to blow its nose and keep the nose clean are always appropriate measures,
- vitamins, trace elements, sulfur (*Rhinathiol, Solacy, and Oligosols*, etc.) have been sufficiently discussed, but their safe prescription has a positive psychological effect on the family,
- iron deficiency (very common) correction,
- removal of the vegetations remains the most effective treatment,
- gastro-esophageal reflux may require specific treatment.

The condition heals spontaneously around the age of 6 to 7 years without any significant consequences, at least in uncomplicated forms.

3. Angina

3.1. Etiology

Angina and acute tonsillitis are acute inflammations of the palatine tonsils.

They occur readily in children and young adults (rarely in babies under 18 months of age), but may also be observed in adults at any age.

Depending on the patient's age, 50 to 90% of angina cases are of viral origin (adenovirus, *Influenza* virus, respiratory syncytial virus, and *para-influenza* virus, etc.).

Among the bacterial agents responsible for angina, Group A b-hemolytic streptococcus is often the first to be found (20% throughout the age groups). Angina caused by Group A b-hemolytic streptococcus only represents between 25 to 40% of angina cases in children and 10-25% in adults. It occurs primarily from the age of 3 years onwards, with an incidence peak in children aged between 5 and 15 years. The condition is rare in adults.

Group A b-hemolytic streptococcus angina usually improves within 3 to 4 days, even in the absence of treatment. However, these infections can give rise to potentially serious complications (post-streptococcal syndromes such as acute articular rheumatism [AAR], acute glomerulonephritis [AGN], and local or general septic complications), for which prevention justifies the use of antibiotic therapy.

Due to the inherent risk of Group A b-hemolytic streptococcus infection, and because antibiotics are useless in viral angina cases, only patients suffering from Group A b-hemolytic streptococcal angina can justifiably receive antibiotic treatment (except for highly occasional *Corynebacterium diphtheriae*, *Neisseria gonorrhoeae*, and anaerobic bacterial infections, which have a different clinical picture).

3.2. Clinical diagnosis

Angina is an inflammation caused by infection of the tonsils, or the entire pharynx. It is a syndrome that combines :

- fever;
- painful discomfort when swallowing (odynophagia);
- changes in the appearance of the oropharynx.

At times, other symptoms may be observed: abdominal pain, rash, and respiratory symptoms (runny nose, coughing, hoarseness, and breathing difficulties). These symptoms are variously associated and variable depending on the causative agent and age of the patient.

Clinical examination of the oropharynx confirms the diagnosis of angina; there are several possible aspects :

- in the vast majority of cases, the tonsils and pharynx are congested: erythematous angina;
- the condition can be combined with an abundant purulent coating that covers the surface of the tonsils: erythematopultaceous angina;
- the pharynx may present vesicles: vesicular angina;
- ulcerative angina and pseudomembranous angina are rarer and should evoke a specific etiology: Vincent's disease, infectious mononucleosis, or diphtheria;
- concomitant sensitive lymph nodes are often present.

However, the aspect of the oropharynx is not predictive of Group A b-hemolytic streptococcal angina. This latter can indeed be erythematous, erythematopultaceous, or even unilateral and erosive. Clinical symptoms may facilitate the diagnosis of Group A b-hemolytic Streptococcus (GAS) angina, but their predictive value is often insufficient (Table 1) :

- fever >38°C;
- presence of exudate;
- painful cervical adenopathies;
- no coughing.

Equally, clinical scores have been proposed, taking into account four items :

Table 1. Etiological orientation elements for angina (source: ANAES)

	Group A b-hemolytic streptococcus angina	Viral angina
Epidemiological	Epidemic - winter and the beginning of spring Age: incidence peak between 5 and 15 years old (possible occurrence from 3 years onwards)	
Functional or general signs	Brutal beginning Intense dysphonia No cough High fever	Progressive beginning Moderate or no dysphonia Presence of cough, rhinitis, hoarseness, diarrhea, arthralgia, and myalgia
Physical signs	Intense pharyngeal erythema Purpura on the palate Exudate Sensitive adenopathy satellites Scarlatiniform rash	Vesicles (coxsackie, herpes) Rash suggestive of a viral disease (for example hand, foot and mouth disease [HFMD]) Conjunctivitis

Each item is worth 1 point, resulting in a score of 0 - 4. Accordingly, a score of 1 indicates a 5% probability of GAS infection. Such a score, notably in adults, enables the decision not to prescribe antibiotics. However, the predictive value of the score is often insufficient.

Generally, due to the inherent risks of GAS infection, the identification of these forms of angina impacts the therapeutic approach chosen.

In cases of erythematous or erythematopultaceous angina, as no clinical sign or score exhibits a positive or negative predictive value in confirming the streptococcal origin of the angina (except for typical cases of scarlet fever), *only microbiological confirmation tests enable the practitioner to identify patients with GAS angina.*

3.3. Bacteriological diagnosis

Rapid diagnostic tests (RDTs), which can be carried out by the practitioner, are recommended. In the laboratory, these tests have a similar specificity to those of cultures, and a higher sensitivity at 90%. Following the collection of an oropharyngeal sample, these tests allow the wall antigens (M protein) of *Streptococcus pyogenes* to be observed (taxonomic name for GAS) following extraction. The results are available in approximately 5 minutes.

In infants and children under 3 years of age, RDTs are commonly pointless, as the angina seen in this age group are generally viral, and only very occasionally caused by streptococcus.

RDTs are recommended in all patients with erythematous or erythematopultaceous angina :

- a positive RDT result confirms the GAS etiology and justifies the prescription of antibiotics;
- a negative RDT in a subject with no acute articular rheumatism (AAR) risk factors does not justify any additional systematic testing by culturing or antibiotic treatment. Hence, only analgesic and antipyretic agents are useful.

Certain rare circumstances (occasional in main land France) evoke a context of AAR risk, these include :

- medical history of AAR;
- age between 5 - 25 years, accompanied by a previous history of multiple GAS angina episodes or visits to regions where AAR is endemic (Africa or French overseas department and region), and possibly certain environmental factors (social conditions, sanitary and economic conditions, promiscuity, and a closed community).

In the context of AAR risk, a negative RDT can be confirmed by culturing. If the culture result is positive, antibiotic treatment is initiated.

3.4. Clinical forms

Numerous other bacteria can be found in throat samples of patients suffering from angina. Certain species have no pathogenic role and are commensal: *Haemophilus influenzae* and *para-influenzae*, *Branhamella catarrhalis*, pneumococcus, staphylococcus, various anaerobic germs, etc. Others have a minor pathogenic role, such as Groups C, G, E, and F streptococcus, gonococcus (adults, epidemiological context), and *Arcanobacterium haemolyticum*, whereas *Corynebacterium diphtheriae* is rarely responsible for angina in France.

These bacterial germs :

- either only rarely give rise to complications: Groups C, G, E, F streptococci and *Arcanobacterium haemolyticum*;
- or are not sensitive to penicillin and do not grow on the culture medium used for angina, such as: gonococcus, *Arcanobacterium haemolyticum*, and *Corynebacterium diphtheriae*. In other words, neither systematic treatment with penicillin nor systematic throat samples are suitable for patient

screening and treatment;

- or exhibit a context or clinical symptoms that are sufficient to require testing and appropriate treatments (ulcerative necrotic angina, false-membrane angina, etc.).

With the exception of diphtheric angina, gonococcus angina, or necrotic angina caused by anaerobic germs (Vincent's disease) that all justify adapted antibiotic therapy, no study has confirmed the usefulness of antibiotic treatment in the following cases :

- viral origin angina;
- b-hemolytic streptococcal angina that does not belong to Group A.

Depending on the appearance of the oropharynx, various etiologies are evoked.

3.4.1. Acute catarrhal angina (or erythematous angina)

Elles sont le plus souvent d'origine virale, peuvent inaugurer ou accompagner une maladie infectieuse spécifique : oreillons, grippe, rougeole, rubéole, varicelle, poliomyélite...

Enfin, une angine rouge peut constituer le premier signe d'une scarlatine, maladie infectieuse d'origine microbienne. Une température à 40 °C avec vomissements, l'aspect rouge vif du pharynx, des deux amygdales et des bords de la langue, l'absence de catarrhe rhinopharyngé doivent faire rechercher un début de rash scarlatineux aux plis de flexion et pratiquer un frottis amygdalien pour mettre en évidence un streptocoque β -hémolytique A.

Fig 5.



3.4.2. Angina with purulent exudate (or erythematopultaceous angina)

These forms often succeed the previous one and are characterized by the presence of bright-red tonsils with purulent exudate: of yellowish gray, with spots or streaks, thin and brittle, easily dissociated, and not exceeding the tonsillar area. Functional symptoms are generally more noticeable. In addition to viruses and GAS angina, the causal agent may be hemolytic streptococcus other than Group A, staphylococcus, pneumococcus, *Pasteurella tularensis* (tularemia), or *Toxoplasma gondii* (toxoplasmosis).

Fig 6.



3.4.3. Pseudomembranous angina (or false-membrane angina)

Examination of the pharynx shows false membranes with a pearly sheen that can extend beyond the tonsil region onto the uvula (uvula palatinae), soft palate, and its arcus palatini.

It is necessary to think particularly of infectious mononucleosis (Epstein-Barr virus) when angina is prolonged and associated with diffuse adenopathies, splenomegaly, marked asthenia, and purpura of the soft palate. A complete blood count (hyperleukocytosis with hyperbasophilic mononuclear cells) and infectious mononucleosis serology testing confirm the diagnosis. Treatment is symptomatic.

Diphtheria, formerly a classic etiology, has become rare in France ever since compulsory vaccination. However, one must always keep this etiology in mind when confronted with rapidly extensive pseudomembranous angina, associated with unusual pallor and fatigue. The false membranes are sticky and non-severable. With a growing population of transplant patients, two factors guide the diagnosis, notably the lack of vaccination as well as return visits to an endemic area. Isolation (for 1 month), diphtheria serotherapy (10,000-20,000 U in children; 30,000-50,000 U in adults), and screening for *Corynebacterium diphtheriae* must be implemented immediately in order to avoid malignant forms, previously of very poor prognosis. Antibiotic therapy must be used in combination.

Other possible but rare causes include staphylococcus, streptococcus, pneumococcus, or other mononuclear syndromes (CMV and HIV).

If in doubt, a diphtheria serotherapy and antibiotics are instituted immediately.

Fig 7.



3.4.4. Ulcerative and necrotic angina

Ulceration, usually unilateral, is deeper and covered by a necrotic coating.

Vincent's disease starts develops insidiously in a teenager or young adult in a generally poor state (fatigue, overworked during examination times, etc.)

- general and functional signs are minor: sub-febrile state, mild unilateral dysphagia, followed by bad breath;
- examination reveals a grayish-white purulent coating on the tonsil that is crumbly, covering a slow developing ulceration with irregular and raised edges, which is non-indurated to the touch. Lymph node reaction is minimal;
- throat swab shows a fusospirochetal association. The blood count result is normal;
- there is often an oral starting point (gingivitis, dental caries, or lower wisdom tooth pericoronitis);
- evolution is benign in 8-10 days. The main differential diagnosis is tonsil cancer;
- treatment with penicillin (after having eliminated syphilis) is very efficient and results in a speedy recovery.

Syphilitic chancre sores on the tonsils have a very similar appearance, however:

- ulceration of the tonsil is unilateral, on a telltale sign induration;
- adenopathy is more pronounced, with a large central lymph node surrounded by smaller lymph nodes;
- throat swab samples with ultramicroscopic examination reveal *Treponema pallidum*.

Moreover, the anamnesis is often delicate. Syphilitic serology testing confirms the diagnosis (initial testing and at Day 15): VDRL is positive 2-3 weeks following chancre, TPHA is positive 10 days after chancre, FTA is positive very early (7 to 8 days) and has excellent specificity, whereas the Nelson test is positive much later at 1 month. HIV serology testing is systematically proposed. Penicillin therapy is the standard treatment, such as *Extencillin* (2.4 MU at 8-day intervals) or *Biclinocillin*.

Fig 8.



3.4.5. Vesicular angina

Vesicular angina is characterized by exulceration of the epithelial layer, followed by an elusive vesicular rash on the tonsils and arcus palatini.

Herpes angina is a prime example, which is usually caused by Type 1 *Herpes simplex* virus:

- onset is brutal with a temperature of 39-40°C, along with shivering and intensely painful dysphonia;
- in the first few hours, small clusters of hyaline vesicles are seen on the bright-red tonsils, while later in the course of the disease, white exudate spots arise surrounded by a red ring, converging into a false membrane of polycyclic contours. This exudate covers the superficial erosions with well-defined edges;
- labial or nostril herpes is frequently associated;
- the evolution is benign in 4 to 5 days, without complications or sequelae;
- treatment is only symptomatic.

Herpes angina has a very similar symptomatology and is caused by Group A coxsackie viruses, occurring mainly in young children. Its evolution is also benign, and treatment is symptomatic.

Fig 9.



3.4.6. Gangrenous, necrotizing angina

This angina, caused by anaerobic germ infections, occurs in patients in a very fragile state: diabetes, kidney failure, and blood diseases. It is mainly of historical interest.

3.5. Treatment

The prescription of antibiotics in GAS angina has several objectives :

→ ***When should treatment be initiated?***

Initiation of treatment can be immediate or postponed to the 9th day of onset of symptoms, as the antibiotic efficiency relative to AAR prevention is maintained in the latter case. This observation allows time for diagnostic investigations to be performed and antibiotic treatment to be tailored to each situation.

→ **Which treatment should be provided?**

Penicillin is the standard therapeutic agent for angina, being the only one to have demonstrated direct effectiveness in AAR prevention.

- b-lactams, treatment duration of 10 days :
 - penicillin V (for example, *Oracillin*);
 - ampicillin, including bacampicillin (for example, *Penglobe*) and pivampicillin (for example, *Proampi*);
 - oral first-generation cephalosporins (cefaclor, cefadroxil, cefalexin, cefatrizine, cefradine, and loracarbef).
- Shortened treatment period (validated by AMM), *which is preferred in order to improve compliance* :
 - amoxicillin (for example, *Clamoxyl*): 6 days;
 - cefuroxime-axetil (for example, *Zinnat*): 4 days;
 - cefpodoxime-proxetil (for example, *Orelox*): 5 days;
 - cefotiam-hexetil (for example, *Texodil*): 5 days.

Macrolides are only used as an alternative to b-lactam treatment, notably when the latter is contra-indicated, particularly in cases of hypersensitivity. This restricted prescription is due to the risk of macrolide-resistant streptococci.

- Macrolide treatment duration of 10 days :
 - dirithromycine (for example, *Dynabac*);
 - erythromycin (for example, *Erythrocin*);
 - roxithromycine (for example, *Rulid*);
 - spiramycin (for example, *Rovamycine*).
- Shortened treatment period (validated by AMM) :
 - azithromycine (for example, *Zithromax*): 3 days;
 - clarithromycine (for example, *Zeclar*): 5 days;
 - josamycine (for example, *Josacine*): 5 days.

The combination of amoxicillin-clavulanic acid (for example, Augmentin) and cefixime (for example, Oroken) is no longer indicated for angina.

It is recommended to inform the patient of the following :

- treatment with antibiotics should be limited to GAS-induced angina (except for rare diphtheric, gonococcal, and anaerobic germ angina);
- need to respect posology (dose and number of administrations per day) and length of treatment.

Symptomatic treatment aimed at relieving discomfort is notably based on antalgic and antipyretic agents. However, due to their associated risks, neither non-steroidal anti-inflammatory agents at anti-inflammatory dose levels nor systemic corticosteroids should be prescribed.

It is not recommended to administer antibiotics to a patient as a preventative measure. The persistence of symptoms after 2 - 3 days should result in reexamination of the patient.

3.6. Differential diagnosis of angina

Especially at the early stages or during superficial examination, angina may be confounded with the following:

Tonsillar cancer

The absence of common infection signs, patient's age, unilateral affliction, severe induration and bleeding when touched, as well as lymph nodes of a malignant nature lead to a biopsy being taken, which is the key to diagnosis. Tonsillar cancer should be systematically evoked.

Buccopharyngeal symptoms of hemopathy

- As a consequence of neutropenia: pure agranulocytosis, of drug origin, toxic, idiopathic, etc.
- pseudomembranous lesions are disseminated on the whole pharynx region with rapid expansion;
- lesions do not bleed and are not sore. There is no adenopathy;
- complete blood count and myelogram reveal agranulocytosis without alterations of the other bloodlines.
- acute leukosis: tonsillar involvement is associated with hypertrophic gingivitis. Its necrotic evolution and hemorrhagic tendency should lead to a complete blood count and myelogram being carried out in order to ascertain the diagnosis.

Pharyngeal zona

Due to glossopharyngeal nerve involvement, this condition is rare and characterized by its strictly unilateral vesicular rash, which is located on the soft palate, upper third of the arcus palatini, and hard palate, while respecting the tonsil region.

Aphthosis

While aphthosis specifically involves the gingivobuccal mucosa, it can also be found on the soft palate and arcus palatini. It is characterized by one to several crescent or pinhead shaped ulcers that are yellowish in color and very painful. They can be observed in the context of Behçet's disease.

Bullous rashes

These are rare disorders and mainly concern the field of dermatology: pemphigus, dermatitis herpetiformis (Dühring's disease), etc.

Myocardial infarction

The clinical picture may mimic acute angina, because of severe unilateral tonsil pain. There is no general infectious syndrome. Throat examination is normal. The ECG is the key element for the diagnosis.

4. Chronic tonsillitis

Chronic infection of the palatine tonsils exhibits different manifestations in children and adults.

4.1. Chronic tonsillitis in children

Chronic tonsillitis in children is secondary to a local immunological disturbance during the first few years of life and can be favored through inappropriate antibiotic treatments.

Chronic tonsillitis in children is clinically manifested by :

- recurrent angina, often associated with white exudates, which are prolonged, with significant adenopathies and long-lasting asthenia;

- persistence of these angina types:
 - inflammatory condition of the tonsils, hard, atrophic or sluggish, releasing a cloudy or purulent fluid when pressure is applied;
 - inflammatory biological syndrome: hyperleukocytosis and raised C-reactive protein (CRP) levels;
 - chronic cervical and sub-angulo-maxillary lymph nodes;
 - lack of efficiency of potential systemic antibiotic therapy.

The evolution is desperately chronic, resulting in height-weight development delay and school retardation through absenteeism, while promoting local and regional complications (nasal sinus area, otitic, and tracheobronchial) or general complications.

Differential diagnosis: chronic tonsillitis should not be confounded with a simple constitutional tonsil hypertrophy or reactive hyperplasia (infectious disease or allergic reactions). These hypertrophies have no functional impact, meaning that no therapeutic sanction is needed (except possibly in cases of breathing discomfort and sleep apnea caused by mechanical obstruction when the hypertrophy is marked).

Treatment of chronic tonsillitis: tonsillectomy.

4.2. Chronic tonsillitis in adults

Chronic tonsillitis in adults is characterized by a significant scarring of the tonsils, which adds to the normal regression of the lymphoid tissue.

The symptomology, mainly local, is usually moderate, arising more easily in patients who are anxious, dystonic, and cancer phobic. Symptoms include intermittent unilateral dysphagia with otalgia, bad breath, fetid fragmented sputum, and tickly coughing. There are no general signs of infection.

Upon examination, the tonsils are small, encrusted in the arcus palatini, with pockets filled with caseum, scar nodules felt on palpation, as well as yellowish cysts due to crypt occlusion.

Evolution is chronic, yet often benign. Search for and therapeutic management of gastro-esophageal reflux disease may improve evolution.

However, local (intra-amygdala abscesses or peritonsillar abscess) or general complications may occur, and it is standard practice to screen for chronic tonsillitis in the context of a check-up for kidney disease or infectious rheumatism.

Differential diagnosis :

- mainly chronic pharyngitis, where inflammation is widespread across the pharynx, especially in relation to a general disease (diabetes, gout, allergies, etc.), digestive disorder, mycosis (after prolonged antibiotic therapy, chemotherapy, etc.), or long-term use of atropinic agents (antihypertensive drugs, tranquilizers, etc.);
- pharyngeal paraesthesia, phobic manifestations relative to the pharyngeal area: sensation of a foreign body or having a "lump in one's throat" (*globus hystericus*) in a neurotic cancer phobic patient. Local examination is normal.

However, the examination must always be performed very carefully so as not to miss early-stage tonsil cancer, hidden in clefts or behind the arcus palatini. Palpation of the amygdala is an essential gesture.

The treatment consists of minor local means: gargling, surface spraying using a laser, radiofrequency therapy, and cryotherapy. Tonsillectomy is indicated in patients presenting complications.

5. Complications of tonsillar infection

Complications are due to GAS infection and seen during acute angina or during reactivation of chronic angina. GAS-induced angina often evolves favorably in 3 to 4 days, even in the absence of treatment, but infection may give rise to septic, local, or general complications, as well as post-streptococcal syndromes (including AAR and acute glomerulonephritis).

5.1. Local complications

Local and regional suppurative complications in relation with GAS infection are mainly represented by peritonsillar phlegmon, but may include suppurative cervical adenitis laterocervical abscess), retropharyngeal abscess, acute otitis media, sinusitis, mastoiditis, and cervical cellulitis.

→ *Peritonsillar phlegmon*

A peritonsillar phlegmon is a suppurative cellulitis occurring between the tonsil capsule and pharyngeal wall.

The clinical picture is characterized by acute angina or reactivation of chronic tonsillitis, with unusual disease progression :

- temperature remaining in the vicinity of 38°C, and then further rising;
- painful dysphagia, increasing in severity, being unilateral, with painful earache.
- The appearance of the patient is evocative, at the full stage of the disease:
- pale and motionless, head tilted to the affected side of the body, allowing saliva that cannot be swallowed to flow from the mouth;
- bad breath as well as nasal and deaf-like voice.

Examination, which is often rendered difficult by a trismus that blocks the opening of the mouth, reveals edema and then, unilateral arching of the anterior arcus palatini; the tonsil extrudes towards the interior; pharyngeal isthmus is asymmetric, with uvula edema (Photo 3).

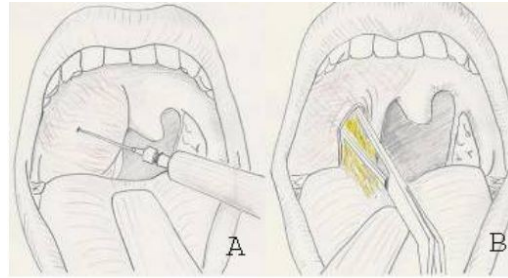
Treatment :

- at the presuppurative stage of phlegmonous angina, antibiotic treatment may induce healing;
- at the phlegmon stage, reflected by insomnia, trismus, edema of the cion (uvula palatinae), and possibly confirmed by exploratory fine-needle aspiration puncture (revealing the presence of pus), surgical evacuation of the suppurative collection is indispensable: vertical incision of the anterior arcus palatini, followed by a clamp debridement (Figure 4.1). At times, a simple aspiration the collected liquid on a daily basis may prove sufficient. Antibiotic treatment is systematically associated.

Fig 10.



Fig 11 : Surgical treatment of right peritonsillar phlegmon



A: Right peritonsillar phlegmon punctured through the anterior arcus palatini, enabling a bacteriological pus sample to be collected (A).

B. The phlegmon is incised through the anterior arcus palatini; the opening is enlarged using a clamp in order to facilitate the evacuation of the purulent liquid (with bacteriological sampling).

Recovery is obtained within a few days.

In order to avoid new episodes, cold tonsillectomy is proposed and should be conducted within a minimum period of 5 to 6 weeks after the phlegmon, but this intervention is no longer performed systematically.

→ **Suppurative cervical adenitis (or laterocervical abscess)**

This is a suppuration of a lymph node within the jugular-carotid chain. This complication is more rare.

Following an angina phase, a painful torticollis and deep cervical thickening with febrile syndrome occur. Imaging (scanner) facilitates topographic diagnosis.

The treatment is based on antibiotic therapy at the pre-suppurative stage. During the swelling phase, the collection is evacuated via cervical incision and drainage.

Fig 12.



5.2. General complications

General complications, which are mainly renal, articular, and cardiac, are related to b-hemolytic Streptococcus A.

The pathogenesis, which has been discussed at length, is likely based on immune mechanisms. These complications are thought to be consecutive to the release of immune complexes, associating b-hemolytic Streptococcus A antigens and IgG immunoglobulins, which are deposited mainly in the renal glomeruli and joints, triggering complement activation and inflammatory reactions.

→ **Acute glomerulonephritis**

Acute glomerulonephritis is often edematous or hematuric, occurring 10 to 20 days after streptococcal angina. While the evolution is generally favorable in children, acute glomerulonephritis can lead to

irreversible kidney failure, especially in adults.

→ **Acute articular rheumatism and post-streptococcal syndromes**

These conditions start 15 to 20 days following the initial tonsil infection:

- either in a brutal and eloquent way by polyarthritis;
- or insidiously in the case of inaugural, moderate myocarditis.
- There is an inverse relationship between the severity of joint damage and the risk of developing cardiac dysfunctions.
- Articular manifestations are more frequently encountered:
- the typical clinical form, now rare, is characterized by migrating, transient, polyarthritis of the large joints. The joint is the site of pain limiting mobility, redness, warmth, and swelling;
- this form is currently replaced either by simple arthralgia, or by a mono-arthritis, which may evoke the diagnosis of purulent arthritis. The spontaneous duration of the rheumatic episode is about 1 month. It disappears without sequelae, while other areas may be affected, with no systematic order.

Cardiac manifestations make up the essential prognostic element :

- their prognosis is both immediate, involving the risk of heart failure, and delayed, involving the risk of valvular sequelae. They are even more common as the subject's age decreases. Cardiac involvement may be either an isolated impairment or involve all three cardiac layers. Cardiac ultrasound is instrumental in confirming the diagnosis and monitoring disease progression;
- endocardial involvement is the most serious condition. At the beginning, it is detected via a heart murmur related to cardiac insufficiency, which is of mitral rather than aortic origin. At a later stage, murmurs related to mitral and aortic stenosis may occur;
- myocardial involvement is reflected by the appearance of heart failure signs, which are of very poor prognosis. Rhythm, repolarization, and conduction disorders are common and evocative. On chest x-ray, the heart's size is increased;
- pericardial involvement, which is very uncommon, is suspected in the presence of precordial pain, pericardial friction rub sounds, increased cardiac size on imaging, or repolarization abnormalities on ECG.

Skin manifestations :

- Meynet nodules are exceptional: subcutaneous, firm, painless, ranging from a few millimeters to 2 cm, these nodules occur next to bone surfaces and tendons, especially near the elbows, knees, wrists, and ankles. They last for 1 to 2 weeks;
- Erythema marginatum has a fleeting evolution, and is characterized by pink macules that are non-pruritic and found on the limb roots and on the trunk.

Neurological manifestations: Sydenham's chorea should be evoked in the presence of involuntary, disordered, anarchic, diffuse, and bilateral movements. Similarly to valvular stenosis, these neurological symptoms occur only following numerous inflammatory relapses.

General manifestations: fever is very common and non-sustainable, responding well to anti-inflammatory drugs, including non-steroidal anti-inflammatory agents. Abdominal pain, linked to mesenteric adenolymphitis or cardiac liver, is observed in 5-10% of cases. There is hyperleukocytosis, and inflammatory markers are raised, with sedimentation rate levels exceeding 100 in the first hour.

Curative treatment :

- in major post-streptococcal syndromes: the patient must rest in bed for 3 weeks, under corticosteroid treatment (to limit or prevent cardiac valvular changes, at a dose of 2 mg/kg/day without exceeding 80 mg/day until normalization of the sedimentation rate, followed by gradual reduction) and penicillin V to sterilize the pharyngeal site, which is relayed by subsequent prophylaxis;
- in minor post-streptococcal syndromes: salicylates and penicillin V.

Preventive treatment :

- antibiotic prophylaxis (to prevent any AAR recurrences in relation to GAS pharyngeal infection) is initiated at the end of curative treatment: benzathine penicillin (*Extencilline*) and in case of allergy, a macrolide.
- duration of antibiotic prophylaxis is 5 years in cases of major syndromes, but only 1 year for minor forms. It is recommended to re-initiate preventive treatment when the patient must stay in closed community (station, dormitory, etc.).

6. Tonsillectomy indications

6.1. Indications

Although tonsillectomy is no longer carried out as frequently as it was in the past, the intervention currently has more accurate and indisputable indications :

- For infection-related reasons:
 - In the case of recurrent acute tonsillitis (whereby the frequency and severity of angina leads to a failure to thrive and falling behind at school - three to four episodes per winter, on two consecutive winters, are part of the commonly accepted schemes) or in the case of chronic tonsillitis in children (tonsillitis with local and regional inflammatory symptoms persisting for 3 months or more, and not responding to appropriate, well-monitored medical treatment).
 - In the case of general complications (nephritis, post-angina rheumatism), when the tonsils constitute a streptococcal infectious focal site. The intervention must be covered using antibiotics therapy.
- For reasons related to pharyngeal obstruction:
 - in children, hypertrophy of the tonsils may cause pharyngeal obstruction with snoring at night, breathing difficulties at night with dyspnea, sometimes respiratory pauses, restless sleep with nightmares, being woken during the night, and food blockages. This obstruction may lead to failure to thrive, thoracic deformations, or heart problems;
 - in the adult, hypertrophy can cause or facilitate the syndrome of obstructive sleep apnea, with snoring at night and sleep apnea resulting in morning fatigue, daytime sleepiness, morning headaches, and nocturia. The diagnosis of obstructive sleep apnea syndrome is established using polysomnographic sleep recordings.

6.2. Contraindications

There is no absolute contraindication to adenoidectomy or tonsillectomy.

Relative contraindications should be considered on a case-by-case basis :

- coagulation disorders can, usually, be detected and are not a contraindication when surgery is imperative;
- cleft palates and submucosal divisions must be screened for clinically. They represent a contraindication relative to the adenoidectomy due to the risk of decompensation of potential Velar insufficiency, which could be masked by adenoid hypertrophy. Amygdalectomy is not contraindicated;
- in case of fever (temperature of $>38^{\circ}\text{C}$), the intervention should be postponed for a few days.

Existing allergy and asthma are not considered to be contraindications for adenoidectomy or tonsillectomy.

Recovery is obtained within a few days.

To avoid new episodes, cold tonsillectomy is proposed to be conducted after at least 5 to 6 weeks following the phlegmon, but this intervention is no longer performed systematically.

Points essentiels

- The adenoid vegetations represent a hypertrophy of the pharyngeal tonsil.
- The rhinopharyngitis in children represents an adaptation to the microbial world.
- Rhinopharyngitis is the turning point of infectious disease in children.
- Only the complications associated with rhinopharyngitis justify antibiotic therapy.
- Acute catarrhal angina arising within the context of respiratory tract catarrh is often of viral origin.
- Short-term treatments are an interesting alternative in order to improve therapeutic compliance.
- Infectious mononucleosis must be suspected, regardless of the clinical features of the angina, if angina is associated with polyadenopathy, splenomegaly, and noticeable asthenia.